ART. XVII.—1. *De l'Ataxie Locomotrice Progressive.* Par le Dr. DUCHENNE (de Boulogne). (*Arch. Gén. de Médecine*, Dec. 1858; Jan., Fev., and Avril, 1859.)

On Progressive Locomotor Ataxia. By Dr. DUCHENNE, of Boulogne.

2. *Études Cliniques et Histologiques sur l'Ataxie Locomotrice Progressive.* Par HIP. BOURDON, &c. (*Ibid.*, Nov. 1861; Avril, 1862.)

Clinical and Historical Researches on Progressive Locomotor Ataxia. By Dr. BOURDON.

3. *Erfolge der Behandlung der progressiven spinal Paralyse durch Silbersalpeter.* Von C. A. WUNDERLICH. (*Archiv der Heilkunde*, Apr. 1861.)

Results of the Treatment of Progressive Spinal Paralysis by Nitrate of Silver. By Prof. WUNDERLICH.

4. *Weitere Erfahrungen, &c.* (*Ib.*, Dec. 1862.)

Further Results of Experience in the Treatment, &c. By the same.

5. *Ueber degenerative Atrophie der spinalen Hinterstränge.* Von Prof. Dr. N. FRIEDREICH, in Heidelberg. (*Virchow's Archiv*, xxvi., 391, 433, and xxvii., 1.

On Degenerative Atrophy of the Posterior Columns of the Spinal Cord. By Prof. FRIEDREICH.

6. *De l'Ataxie Locomotrice.* Par GEORGE DUJARDIN-BEAUMETZ. (Paris, 1862, 8vo. pp. 73.)

On Locomotor Ataxia. By Dr. DUJARDIN-BEAUMETZ.

THE intimate relations of physiology and pathology as the scientific groundwork of practical medicine have nowhere been more beautifully illustrated than in the advancement which has been made, of late years, in our knowledge of diseases of the nervous centres, and especially of the spinal cord. In spite of the discoveries of Sir Charles Bell, the interpretation of the phenomena presented by diseases of this organ, was neither clear in itself nor fruitful of practical results in diagnosis and therapeutics. The treatises on practical medicine did not successfully attempt to establish sharply defined symptomatic species, nor even endeavour to found a classification of those which they admitted upon determinate anatomical lesions. In describing the latter, pathologists seemed to ignore the multiple functions which had been proved by experiment to belong to the spinal marrow, and the lesions of this organ were recorded with little attention to the independent offices of its several parts. This is the more singular when it is considered that numerous cases, long ago placed on record, which, as will be shown in the sequel, abundantly demonstrated the co-existence of at least one special disease of the spinal marrow and the peculiar alteration of this organ which constitutes its anatomical character. To experiments on living animals, against which an unreasoning susceptibility every now and then pricks the knight-errants of humanity to run a muck, the world owes this most important advancement of medical knowledge. Fortunately for society, as well as for the progress of the healing art, the chief pioneer in these researches has been guided and restrained within just limits by his

acquaintance with disease and his office as a physician. At every step in his experiments, Dr. Brown-Séquard has paused to compare the phenomena created by experiment with those evoked by disease, and reading the one in the light of the other, has reached results which, whatever may be the ultimate formula in which they shall be expressed, are sure even now to embody more and more exact truth than any arrived at by a single pathway of discovery.

To take a review of the several forms of paralysis in which the various functions of the spinal cord are deranged, impaired, or lost, would require a volume instead of the few pages to which we are at present restricted. But it may be remarked, in general, that while very much remains to be done, a great deal more has been already accomplished than is commonly recognized in systematic treatises towards determining the multiple causation of paralysis, and in proving that in very many cases the spinal axis reflects the operation of influences derived from remote organs, while itself remains essentially sound. This has been amply demonstrated by the eminent teacher we have named, in a course of lectures delivered in 1859,[1] and by E. Gubler, in a series of articles published in 1860–61.[2] All of the other forms of paralysis have been within quite a recent epoch more narrowly examined than ever before, including that of the insane, of drunkards, paralysis from various poisons, from muscular atrophy, &c., as well as those resulting from acute diseases of the spinal cord and its membranes. Leaving these out of consideration, we propose to confine our attention to a review of the present state of knowledge regarding *locomotor ataxia*, as it has been called in reference to its peculiar symptoms, or *tabes dorsalis*, as it has been more significantly termed in view of its anatomical characters. A somewhat more expressive title borne by the same affection is *myelo-phthisis*.

This disease has been defined by Duchenne as "a progressive loss of the co-ordination of muscular movements, and apparent paralysis, although the muscular power remains unimpaired;"[3] and this definition is accepted as his own by Trousseau.[4] It would have been in accordance with the results of observation to add that the disease first manifests itself in the lower extremities. This is strikingly shown in the interesting group of cases observed by Dr. N. Friedreich, and in those collected by him in the historical notice which he has furnished of the observations of other physicians.[5] The earliest symptom of the affection consists of a sense of weakness and weariness in both legs together, or at first in one alone. It would appear from a case related by Lecoq,[6] that the characteristic peculiarity of movement may, for a long time, or even permanently, be confined to a single leg. His patient could readily walk backwards, or go up and down stairs, but in ordinary progression the left leg was quite uncertain in its movements. After a long time, in all cases, it may sometimes be after several years, and when the patient is no longer able to maintain an erect position a similar defect of co-ordinating power may involve the upper limbs. In one of Friedreich's cases the leg and arm of the same side were simultaneously attacked, while a sense of weakness and weariness affected the back and loins. In general, when one leg only is at first involved, the arm of

[1] London Lancet, April to Sept. 1860.
[2] Archives Générales, 1860–61.
[3] Arch. Gén., Dec. 1858, p. 641.
[4] Abeille Méd., Avr. 1861, p. 126.
[5] Virchow's Archiv, vols. xxvi. and xxvii.
[6] Arch. Gén., Jan. 1861, p. 693.

the same side becomes involved before the leg of the opposite side. In the early stages of the disease the gait is only unsteady like that of a person slightly intoxicated; in going up stairs the patient has a peculiar swinging movement, and even in the standing posture there is a peculiar vacillating or balancing motion of the body. Bourdon attempts to draw a distinction between the gait characteristic of the early stages of this disease and that which is exhibited in some cases of disease of the cerebellum, by comparing the latter to the tottering and reeling movements of a drunken man, while of the former he says the patients walk with difficulty, because the will no longer directs their movements.[1] This distinction does not appear to be a real one. In both cases the will is ineffectual to regulate the muscular movements concerned in walking. A characteristic difference between the gait of true paraplegia and that of locomotor ataxia consists in this, that in the former the limb is thrown forward by an energetic movement of the trunk, when it oscillates for a moment, and then falls heavily to the ground; in the latter, on the other hand, the co-ordinated movements of the lower extremities are suddenly interrupted, or are so irregular as to seem to be no longer under the control of the will.[2] It is, moreover, to be remarked that after years' duration of the disease, and even when the patient has long been confined to bed, or is quite unable to stand erect, he is still able to perform any simple movement of the limbs, as flexion, extension, &c., and that with considerable force. M. Trousseau relates, *apropos* of this symptom, that one of his medical friends begged him to visit a patient who, he said, was affected with a very odd kind of paraplegia. It was an old man of fourscore, who appeared to be in the best of health, except that for a year preceding he had suffered from paralysis of the bladder, and was unable to move from his chair. One day, the physician desiring to estimate the loss of muscular power in his patient's leg, requested the latter to extend the limb as strongly as possible. The experimenter, not expecting any exhibition of muscular power in the leg which he was holding flexed, was much astonished at finding himself thrown violently to the opposite side of the room. M. Trousseau himself was unable to flex or extend the limbs against the patient's will, and the invalid was able, without bending, to carry M. T. on his shoulders. It was evident, says M. T., that the case was one of muscular ataxia, and not of paralysis. It must not be supposed, however, that the muscular force is so perfectly preserved in every case. It is sometimes materially impaired, but never so completely lost as in true spinal or cerebral paralysis.

The real character of the disease is exhibited when the patient endeavours to execute any combined movement. When the upper extremities are affected, if he attempts to grasp anything, many abortive efforts are made for this purpose. A glass or spoon is not steadily directed to the mouth; and threading a needle or buttoning the clothes becomes a very difficult task. By these symptoms one cannot fail to be reminded of chorea. Indeed, the analogy of the motor disturbances in the two affections deserves a closer investigation. In chorea, spasm is superadded to motor ataxia.

Muscular spasm is rarely noticed in this affection, a circumstance which distinguishes it strikingly from most other diseases, whether acute or chronic, of the spinal marrow. Sooner or later, according to Friedreich, the power of vocal articulation becomes impaired, but in general only after the upper

[1] Arch. Gén., Avr. 1862, p. 387.
[2] Dujardin-Beaumetz, De l'Ataxie Locomotrice, Paris, 1862.

extremities have for some time been affected. It gradually grows indistinct and stammering, so that the patient is with difficulty understood. In one case only noted by this author was there complete paralysis of the tongue, and that only for short periods; but in all, with this single exception, the patient could move the tongue in any desired direction. When the organ was thrust out it could not be kept still, but was affected with twitching and trembling. In one case a peculiar incoördination of the laryngeal muscles seems to have occasioned a spasmodic cough with whistling inspiration and dyspnœa.[1] Double vision, strabismus, falling of the upper eyelid, amaurosis, and a peculiar oscillatory motion of the eyeball, have been occasionally observed. The muscles of mastication and deglutition appear never to be affected, and those which control the rectum and the bladder are rarely impaired.

Some discordance is to be observed between the accounts given by different reporters concerning the sensibility of the skin. Friedreich, whose observations bear the stamp of accuracy, declares positively that in all of his cases cutaneous sensibility was perfect, or but very slightly diminished. The lightest pressure upon the surface of the body, as well as strong impressions, such as punctures with a needle, were in all cases perfectly perceived, and the place where they were made correctly indicated by the patients even when their eyes were closed. A similar result followed the use of the test by a pair of compasses opened to a greater or less degree. On the other hand, Dujardin-Beaumetz maintains that the sense of pain is sometimes lost, although he admits that it is less frequently so than the sense of touch, and that it may even be morbidly increased.

This derangement of sensibility is to be distinguished from that of the muscular sense, which is pretty generally admitted to be materially impaired in the greater number of cases; indeed this symptom constitutes one of the most striking phenomena in certain cases of the disease under consideration. It consists of an erroneous judgment of the degree of resistance opposed to muscular efforts. The patient, especially if unaided by his sight, no longer correctly appreciates the force or the purpose of his movements. They either exceed or fall short of their just degree, or are imperfectly harmonized, so that a sort of tremulousness or vacillation characterizes even the most simple movements, which, indeed, are often executed after a series of oscillatory efforts. But, according to Bourdon, as soon as the eye estimates the extent of the required movements, the muscles are competent to execute them.[2] This writer, however, maintains that the disordered movements just described are due to what he designates as "the loss of the sense of muscular activity," while in locomotor ataxia, properly so called, the disorder of muscular movements continues, in great part, at least, even when the sight intervenes to control them. In every attempt at walking, he says, the limbs are thrown forcibly in all directions, and often progression and even standing are impossible. The mere circumstance that the co-operation of vision is sometimes necessary to render muscular movements efficient, does not appear to be a sufficient ground for regarding the cases in which this circumstance obtains as belonging to a separate disease, any more than the degree of muscular ataxia itself. It is true that muscular ataxia does not necessarily imply imperfection of the muscular sense; but the latter has very seldom, if ever, been observed without the former. Future anatomical investigations may perhaps show that the two

[1] Dujardin-Beaumetz, op. cit. [2] Arch. Gén., Apr. 1862, p. 388.

functional disorders in question are each dependent upon special lesions. Meanwhile it is certain that the records of cases do not always clearly distinguish between the two, or even recognize the existence of the separate functions which present them. The one (ataxia), it should be recollected, relates to the performance of combined movements, as standing, directing the foot or the hand to a given point, &c.; the other (loss of muscular sense) relates to the sensations by which the mind judges of the position of the limb, the nature and degree of obstacles opposed to its movements, &c.

Among the subordinate symptoms remaining to be noticed may be mentioned a sense of giddiness which has sometimes been observed in the early and sometimes in the advanced stages of the disease, and which occurred not only in the erect but sometimes in the horizontal posture also. Friedreich did not observe any disorder of the mind or of the special senses. On the other hand, Duchenne affirms that some disorder of vision was met with in seventeen out of twenty cases; and Trousseau speaks of the frequent occurrence of strabismus and double or feeble vision in the early periods of the disease. There appears to be little or no diminution of muscular contractility under electro-magnetic stimulus at any stage; but in cases of many years' duration the electro-muscular sensibility has been found greatly diminished. For example, although the current excited powerful contractions in the legs they caused a relatively slight sensation, and the same current passed through the arms induced severe pain as well as muscular action. The sexual organs in the female are said to undergo very slight impairment of function, the catamenia remaining regular, and the power of conception continuing. In the male, on the contrary, if there appear to be insufficient grounds for the belief formerly current that tabes dorsalis is one of the direct consequences of excessive venery, it is at least certain that when the disease is established the virile powers rapidly decline and ultimately become extinct. The functions of the digestive and uriuary organs present no special symptoms.

The anatomical characters of *myelo-phthisis*—the lesion which produces chronic and progressive locomotor ataxia—may be considered as established. Duchenne, after describing the symptoms of numerous cases of the disease, avowed his ignorance of the anatomical conditions producing it; he even states that in a case of it which proved fatal by intercurrent disease no lesion of the brain or spinal cord was discoverable. Such a statement cannot now be accepted unless it is supported by the results of a microscopical examination made by an expert. On the other hand, M. Bourdon correctly presents the following as the result of post-mortem examination in thirteen cases observed by himself and various other physicians:—

"The posterior columns of the spinal cord and sometimes the cineritious substance also, had undergone degeneration, were of a grayish-yellow colour and semi-transparent; the posterior roots of the spinal nerves were also atrophied; sometimes the optic nerve, and in one case the tubereula quadrigemina had become altered."[1]

Every new contribution to the subject proves this description to be essentially correct, and the whole goes to confirm the proposition announced in 1858 by Dr. Brown-Séquard, and contained in the volume of his lectures published in 1860:—

[1] Arch. Gén., Avr. 1862, p. 405.

"That in cases of alterations limited to the posterior columns, but occupying all their length and thickness, or only the whole of the lumbar swelling, there is an impossibility of standing or walking, depending upon the loss of the reflex action of the limbs; but that in bed, the patients in such cases can move their lower limbs pretty freely."

The reader will, however, better appreciate the nature of the anatomical lesion in question after examining the following summary of the report published by Friedreich relative to three cases which died of an intercurrent disease (typhoid fever) while under his care. In all of them the ataxia had been of many years' duration.

The external surface of the spinal dura mater showed no visible alteration; but in all three cases the finger could detect some fluctuation in the lumbar region, and, on opening the membranes in this part, a considerable portion of clear transparent liquid escaped, while above and below the dura mater lay in immediate contact with the pia mater. The latter had a milky and turbid aspect the entire length of the posterior columns, was of unusual firmness, with difficulty separable from the substance of the cord, and upon its external face was adherent to the inner surface of the dura mater by numerous white bands and threads. The ligamentum denticulatum in its whole length was thickened and of a milky whiteness. The alteration of the pia mater extended somewhat, but in a less degree, over the lateral columns. These lesions sufficiently demonstrate the existence of a chronic spinal meningitis.

The most striking and essential changes were in the substance of the spinal marrow. Even a superficial inspection revealed, in all the cases, a gutter-like depression, of greater or less depth, along the posterior surface of the cord. It was deepest in the lumbar and dorsal regions. On making transverse sections of the cord it was evident that this depression corresponded to the atrophied posterior columns which, even to the naked eye, had a grayish and translucent aspect in striking contrast with the normal appearance of the lateral columns. In two of the cases this altered portion was firmer and tougher than the normal tissue; in the third its consistence was but slightly abnormal. In all three cases the whole of the lumbar portion of the cord, with the exception already stated, was less firm than natural, which, probably, may be attributed to its post-mortem maceration in the effused serum.

These lesions agree with the symptoms in pointing out the lumbar portion of the spinal marrow as the part first affected, and as showing that the disease proceeds thence to the upper portions of the cord.

In two cases the anatomical alteration had slightly invaded the lateral columns in the lumbar region.

In all the natural line of separation between the opposite halves of the organ had almost totally disappeared so far as the posterior columns were concerned; but the cineritious matter offered no evidence of change when examined with the naked eye, or with the microscope. The pons, the crus, and the cerebellum were free from disease.

The microscope showed the same degeneration wherever the grayish aspect of the tissue existed. In place of nerve fibres were seen delicate empty tubes, imbedded in a granular substance, consisting of cells containing several nuclei; and in proportion to the amount of this substance was the loss of the normal nervous fibres. In these no trace whatever of fatty degeneration could be detected. In the lumbar region the destruc-

tive process was so complete that only after searching a long time could any rudiment of a nerve fibre, with a double contour, be perceived. Corpora amylacea were met with in the midst of the elements already mentioned. The microscope revealed no change of structure in the auterior or in the lateral columus, except at some points of their connection with the posterior columns.

The posterior roots of the spinal nerves were atrophied, being thinner, flatter, and harder than natural, but the anterior were unchanged. The atrophy of the posterior roots was most conspicuous in the lumbar region. Their proper nervous constituents, in a great measure, had disappeared, yet the continuity of all the fibres was not entirely destroyed. The sciatic nerves were also in some degree deficient in nervous matter.

Hence, anatomically, the disease is "*a chronic inflammatory process, resulting in the atrophy of the nerve elements, and which, being confined to the posterior column of the spinal cord, begins in its lumbar region and gradually extends thence both upwards and downwards. With this alteration is associated a spinal meningitis affecting the posterior face of the cord, and proportioned in its degree to that of the alteration of the cord itself.*"

It may be interesting to know that in a recent case of the disease, which proved fatal with phthisis, under the care of M. Trousseau, the same lesions were found by the skilful anatomist, M. Sappey. The lumbar portion of the cord was slightly diminished in volume and of a grayish tint within. The posterior roots of the nerves were considerably atrophied; they had lost from two-thirds to three-fourths of their normal volume, were flattened, and no longer white, but reddish-gray. Some of the nerve tubes of these roots retained their integrity; of the rest, the greater number had lost a portion of their medullary contents, and, in some points, were contracted, and a little further on relatively enlarged. In very many the medulla had completely disappeared at different points, and some of the nerve tubes were quite empty.[1] It will be observed that the lesions in this affection present as uniform a character as those which are aeknowledged to be the anatomical character of any disease whatever. They are almost always confined to the lumbar portion of the cord and its dependent nervous roots, or present sufficient evidences in their nature and degree to prove that they began in that region. Instead of extending into the substance of this complex organ, as it does when traumatie in its origin, the softening extends along the parts just indicated, thereby proving their essential organic and functional independence as distinctly as when effects, analogous to the phenomena of the disease, are produced by the scalpel of the physiological experimenter.

The causes of tabes dorsalis are very obscure. There would appear to be some ground for the belief that hereditary tendency may have a share in its production. At least, according to M. Trousseau, although it cannot be shown that this special disease is transmitted from one generation to another, there is proof, at least, that it is apt to occur in families which manifest a proclivity to nervous affections. A stronger proof is furnished by the cases of Dr. Friedreich, all six of which took place in members of two families. Sex does not appear to exert a decided influence in its causation. Some observers have met with more cases among males and others

[1] Arch. Gén., Avril, 1863, p. 490.

among females. It may be remarked that when the affection was more generally than now ascribed to sexual excesses, the male sex was alleged to furnish the most numerous examples of it. But a comparison of recorded cases does not substantiate this view of its causation, and consequently the inference cannot be admitted. Friedreich lays much stress upon the fact that in all of his cases the first symptoms of the disease manifested themselves about the epoch of the maturity of the sexual development, *i. e.*, between the fifteenth and the eighteenth year. Canstatt gives the age between the eighteenth and the thirtieth year as that in which it most frequently commences; and Dujardin-Beaumetz states that "it almost always begins between the thirty-fifth and the fortieth year." These discordant conclusions prove that no general law governs the matter. The same remark is applicable to the duration of the disease. It is quite indeterminate, but usually very long. Of Friedreich's patients three died of typhoid fever in the twelfth, fifteenth, and sixteenth years of the disease respectively; while in three who were still living the symptoms had lasted for nine, eighteen, and twenty years.

Dr. Brown-Séquard, if correctly reported, regards muscular ataxy as only a symptom incident to various lesions of the brain and spinal marrow.[1] The passage we have elsewhere quoted from his lectures would, on the contrary, favour the conclusion we have adopted that degeneration of the posterior columns of the spinal cord is essentially connected with the peculiar disorder of the muscles now denominated ataxia. Wunderlich is not willing to accept the individuality of the disease and its anatomical expression in the lesions of the spinal cord, maintaining it to be questionable whether these lesions instead of being the original cause of the symptoms are not in reality results of the long functional inactivity of the organ. But, it may well be asked, what, then, is the cause of the degeneration affecting the spinal marrow in cases where this is the sole abnormal point of the whole organism? Certainly, the palpable and visible lesion must be preceded by an invisible and inappreciable organic change, which, if anything, has occasioned the peculiar and progressive loss of the equilibrating power in the lower limbs; and it is more logical to suppose this loss of power to depend upon a central change which sooner or later becomes atrophic degeneration, than to assume a causeless loss of function in an organ as the source of its decay. While leaving out of view for the present, then, the degrees of the lesion in question, we feel authorized by the whole clinical and post-mortem history of muscular ataxia, to regard it as an independent affection, whose phenomena with their analogies and differences it is important to learn.

Now it is very certain that derangement of the motor power of the lower extremities and of other parts cannot be the initial phenomenon of the disease in the great majority of cases; it is rather an evidence of its maturity. Several symptoms have been regarded as characteristic of its early stage. Duchenne has called particular attention to loss of sight and strabismus affecting one eye, and usually temporary in their duration. But others have witnessed nothing of the kind. Perhaps they were less minute in their examination; but this is not to be presumed, nor is it probable that such striking symptoms as those alluded to, would escape the notice even of less practised and minute observers than Friedreich, and Wunderlich, and

[1] Med. Times and Gaz., March, 1863, p. 248 and p. 274.

Bonrdon, and the others who have reported cases of this disease. Nor is it in accordance with general observation that darting pains in the lower extremities precede their loss of coördinating power as Duchenne declares. This symptom is undoubtedly met with in a certain proportion of instances (we have, ourselves, observed it most distressingly developed in a well marked case of the disease), but the records prove that it is oftener absent then present. There remains, then, only the peculiarity of the disorder of motility upon which a diagnosis of the affection can be based; and, as it is probable that this may have been of long duration before its increase has become sufficient to attract attention, the conditions attending its primary development may have remained undiscovered. Once, however, noticed, it is probably taken for a paraplegia, *i. e.*, for a loss of *motor power* in the lower limbs, and hence its distinction from true paraplegia should be as clearly as possible ascertained. It is unnecessary to repeat in this place what has been more than once stated, that the pathognomonic sign of tabes dorsalis is an imperfect power of coördination of the muscles of the lower limbs, and exceptionally of other parts. Such is the typical expression of the disease. But it will readily be understood that the lesion of the posterior columns, with or without that of their corresponding nervous roots, and which gives rise to the symptom in question, may not be the sole alteration of the spinal cord. It may be complicated with lesions of the gray matter, and of the lateral, or of the anterior columns; and thus may be occasioned various degrees of hyperæsthesia, anæsthesia, and true paralysis. Dr. Brown-Séquard has demonstrated these propositions experimentally and clinically.[1] Thus it is that his statement referred to in the last paragraph finds its justification. But it does not follow that there is not a disease originating anatomically in the posterior columns of the spinal cord, running its course chiefly in them, and occasioning the symptoms which we have endeavoured to describe in this article. But we shall return to this subject when speaking of the treatment of the disease.

It will not be without interest or utility to consider the most striking differences between the symptoms of other spinal affections and those of tabes dorsalis. Hysterical paralysis is undoubtedly the disorder which most closely resembles it, presenting as it generally does a diminution of the muscular sense, while the power of muscular movement may be but slightly impaired. Nor, when the loss of power is constant in the same muscles, does it appear easy to ascertain the real nature of the case except by employing, as Duchenne advises, cutaneous and muscular faradisation, which, he asserts, will generally cure the one, but not the other affection. In general paralysis of the insane, there is a loss of power in all the muscles when the disease is fully developed, and during the earlier stage a general tremulousness, but the co-ordinating faculty is not impaired. M. Ducheune describes as general spinal paralysis the gradual failure and final loss of the power of muscular movement, with the loss or diminution from the first of the susceptibility of the paralyzed muscles to the electric stimulus, and the ultimate fatty degeneration of these muscles. These examples may serve to indicate the most important points of differential diagnosis in the study of tabes dorsalis; yet it is, after all, sufficient to bear in mind the distinctive sign of the disease given above, the impairment of

[1] Lectures, Philadelphia, 1860, p. 136.

muscular co-ordination, and to exclude all grounds of hysterical paralysis, to render the diagnosis as nearly certain as possible.

There would be little advantage in discussing the value of the long list of remedies which have been used in the treatment of tabes dorsalis. Revulsives and derivatives, general and local baths, electricity, ergot and iron, nux vomica, and cod-liver oil have all been tried perseveringly, and the result has only served to confirm the melancholy conclusion of Romberg:—

"There is no prospect of recovery for patients of this class; the fatal issue is unavoidable; the only consolation that can be offered to those fond of existence is the long continuance of the disease. If, in any case, the busy activity of the physician increases the sufferings of the patient, it is in tabes dorsalis."[1]

Friedreich, also, concludes his short summary of this part of the subject by saying:—

"It is indeed discouraging to observe how, in spite of active remedies, the symptoms gradually but visibly grow worse, while an evident suspension of its rate of progress may be observed when all medicines are discarded and the patient is confined entirely to dietetic measures."

Almost the only persons who have furnished a less discouraging estimate of the value of remedies in this disease are Dr. B. A. Wunderlich, Professor in the University of Leipsic, and those who in imitation of his example have employed nitrate of silver in its treatment. His first publication was made respecting it in 1861,[2] and in this he maintains that no diagnostician in the world is competent to declare beforehand what precise lesion will be found after death in any case of "progressive spinal paralysis," nor in what degree of disorganization, if any, the spinal marrow may be at a particular stage of the symptoms, nor what are the limits of nature's power in restoring the injured structure of the affected organ.

"My cases," he remarks, "appear to me most distinctly to prove that one may be affected for years with progressive spinal paralysis, and already have sunk into the most deplorable condition, without its necessarily following that the nervous matter essential to the impaired functions shall be destroyed. We ought, rather, as my cases show, not to abandon too readily the hope that the integrity of the function may be restored." (p. 197.)

MM. Charcot and Vulpian[3] assume a similar position.

"The gray matter of the cord," they remark, "is usually intact in these cases; the nerve tubes of the posterior columns are alone altered, and their alteration usually consists in this that the medullary matter has disappeared, while its containing tubes are unimpaired. *It is easy to conceive* that such an alteration is susceptible of repair; that the nervous matter may be reproduced in the tubes, and that these being gradually restored to the normal condition, may regain their functions."

Undoubtedly, such a process may be imagined, but there is no proof whatever that it actually takes place.

However this may be, the real and important question relates to the curability of tabes dorsalis by nitrate of silver. We have examined with great care the cases published by the authors above named, and by M. Herschell,[4] M. Moreau,[5] M. Beau, and M. Vidal,[6] and we have not been able to

[1] Syd. Soc. ed., ii. 400.
[2] Archiv der Heilkunde, ii., 193.
[3] Bull. de Thérap., June, 1862, p. 486.
[4] Bull. de Thérap., Oct. 1862, p. 360.
[5] Ibid., p. 373.
[6] Ibid., Jan. 1863, p. 82.

accept their conclusions as demonstrated. In nine cases the symptoms undoubtedly comprised the peculiar aberration of the co-ordinated movements which is characteristic of tabes dorsalis, and they were all, more or less, benefited, some of them wonderfully improved, by the administration of nitrate of silver. But there was a peculiarity in them all which appears to have been overlooked by their reporters, and which distinguishes them widely from those cases of the disease which Friedreich, Bourdon, and earlier writers described, and which, as we are convinced, requires them to be placed in a different category from the latter. *They were all of a rheumatic origin.* In the history of each we read: "the patient lived in a cold and damp room," or "he had the perspiration checked," &c. before the first spinal symptoms made their appearance.

The operation of such causes followed by locomotor ataxia would seem to point out a chronic spinal meningitis as the real disease in the cases referred to, rather than a progressive degeneration of the posterior columns of the spinal marrow. The one affection is not in its nature incurable, but there are no adequate reasons for believing that atrophied or softened medullary matter can ever be replaced by sound tissue. It is further to be observed, in relation to the efficacy of the proposed remedy, that it is claimed, even by M. Wunderlich, to have effected a cure in only one case out of five, while in the remainder more or less improvement was manifested. Yet these were all comparatively recent cases, and one, the most successful, was not of more than three months' duration when it came under treatment. MM. Charcot and Vulpian, after alluding to these circumstances, state that the duration of their cases was much longer, extending to several years, and that they had all been sent to the Salpêtrière as incurable after having been treated by various methods in different hospitals. Surely, in such cases it is in the highest degree probable that the medullary lesion, if any, had already reached its full development. How, then, can we explain that the success in their treatment was relatively greater than in the more recent cases? We cannot refrain from doubting the reality of the pathological condition ascribed to them all, for we cannot but feel surprise that a medicine should be found efficacious in direct proportion to the gravity of the condition it is proposed to cure.

Among the cases reported by some of the gentlemen above referred to, are several whose relations to tabes dorsalis may be questioned upon other grounds than those suggested. In the second case of MM. Charcot and Vulpian the previous history of the patient includes sciatica and hysteria, numbness of the hands and feet, violent pains in the ends of the fingers, intense and continued pain in the cervical and dorsal regions, &c. These are anomalous symptoms in the affection to which they are referred. In Herschell's case also, there were excessive pains and intolerable cramps in the feet, followed by complete amaurosis. The ataxia, too, is described "as a sort of tremulousness whenever the patient endeavoured to grasp anything." These, too, are not characteristic. The same may be said of Duguet's case, which was complicated with epilepsy. One less open to criticism is that of Vidal, in which the exciting cause appeared to be prolonged sexual excesses, and the symptoms, generally, were characteristic; but, again, incontinence of the urine and feces is mentioned, and obtuseness of sensibility to the touch, although friction was painful, symptoms not observed when the disease has existed in connection with alterations of the posterior columns of the spinal marrow.

Without entering further into a critical analysis of these cases, which

would, however, be both interesting and profitable, we shall conclude this portion of our subject by quoting a report of M. Trousseau's remarks in a clinical lecture.

"He appeared to be apprehensive that the experimental use of nitrate of silver, which is now carried on so extensively, is the result of a partiality which clinical observation does not justify. Of four persons in his wards labouring under this disease, not a single one had been benefited by the treatment in question. He referred, however, to a private patient in whose case it had done wonderfully well, and who assured him that it had saved his life. But although he had known it to cause improvement, he had not seen it effect a single cure."[1]

The ultimate conclusions of Wunderlich himself are far from affording a sure ground of faith in the proposed remedy, since he confesses that he is acquainted with cases which improved very considerably under the influence of mere rest and simple baths; a circumstance, by the way, which strengthens the conviction that his cases were not examples of tabes dorsalis at all. Finally, he confesses that he cannot answer for the permanence of the improvement which appeared to follow the use of nitrate of silver. So that the question of its curative action is still *sub judice*.

It would be to undervalue the results which we have thus briefly analyzed were we to deny the advantage or the propriety of using nitrate of silver in cases presenting the peculiar aberration of muscular motion we have been considering. So long as the practical difficulty of determining the precise nature of the symptom exists in any case, and so long as clear indications for other treatment are wanting, it is not only allowable, but required that a remedy whose virtues are so highly and authoritatively set forth, should be tested to the full extent. In the case of a lady under our care, who lost the co-ordinating power of the lower limbs after a slight but depressing attack of pneumonia, but whose muscular vigour in the same parts remained wholly unimpaired, we made use of the nitrate of silver perseveringly after all other remedies had completely failed. It did not appear to improve the symptoms in the slightest degree, and was at last abandoned because it occasioned gastric oppression and heartburn.

It should be mentioned that the dose of nitrate of silver employed by all the physicians to whom we have referred, was essentially the same, viz., one-fifth of a grain given twice a day at first, and subsequently three times a day. This dose was seldom exceeded, but it was, with short intermissions, continued for many successive weeks. Its good effects appear to have been visible within a few days after its use was commenced.

It will interest reflecting readers to learn by what steps a disease which must always have existed, came at last to be recognized as symptomatic of a definite lesion of the spinal cord. The interest will not be diminished by knowing that so eminent a practitioner and pathologist as Lebert has not described it either in his magnificent work on pathological anatomy, or in his treatise on the practice of medicine. This will seem the more remarkable when it is known that Cruveilhier's Pathological Anatomy, which Lebert's was intended to complete according to the later advances of the science, contains several instances, described at length, of the specific lesions and symptoms of tabes dorsalis. The very first case which he relates was of seventeen years' duration, and, although complicated in

[1] Bull. de Thérap., April, 1863, p. 315.

several respects, it presented the peculiarity that the patient could move her limbs freely, but not stand on them. Another case, of which a detailed account is given, bears this descriptive title. "Complete loss of sensation and only partial of motion in the lower limbs. Yellowish-gray transformation of the posterior columns of the spinal cord." She was able to move her limbs freely in bed, but could not stand. The posterior columns in the dorsal lumbar regions were alone affected.[1]

In Baly's translation of J. Müller's *Elements of Physiology*, Part III., published in 1837, we find the following passage at p. 807 :—

"The cord is always charged, as it were, with motor power, and, although in transmitting the nervous fibres from the brain, it acts as a conductor of the oscillations originating in the sensorium commune, still the intensity of the action excited depends not merely on the strength of the will, but also on the amount of motor power accumulated in the cord. Hence this part of the central organs may retain its property of conducting the volition from the brain, but lose the second power by which it determines the strength of our movements; and this is what happens in tabes dorsalis, a disease caused only by debauchery, and attended with atrophy of the cord. *Here no muscle of the lower extremities is at first paralyzed; all obey the influence of the will, even in the advanced stages of the disease; the patient can execute every movement*, and it is evident that the spinal cord is still unimpaired as a conductor of the oscillation or current originating in the sensorium. But the force of the movements is lost; the patient can neither stand nor walk long at a time; and the power gradually diminishes until the paralysis is complete. This kind of paralysis must be carefully distinguished from others, in which the conducting property of the cord is injured at one point, and the muscles which receive their nerves from below that point are no longer subject to volition, while all other muscles retain their full power of motion."

In this extract the distinction is plainly drawn between a loss of what is now called the co-ordinating function of the cord, and its function as a medium for the transmission of motor power.

It is unnecessary to quote at length the description by Romberg of the disease in question, as it is contained in the Sydenham Society's edition of his work on Diseases of the Nervous System. The symptoms which he attributes to tabes dorsalis are clearly derived from cases in some of which the lesion was composite; but in describing the post-mortem lesions in certain cases, he says :—

"It is a point of especial interest to observe that the posterior, sensory roots [as they were then exclusively considered] are occasionally alone affected in conjunction with the posterior columns of the spinal cord, the anterior motor columns and nerves retaining their normal structure."

Romberg lays peculiar stress upon the assistance which the patients derive from the sense of sight in regulating their movements; indeed, he denominates it a pathognomonic sign.

In 1840 Dr. Brach, of Cologne, wrote an article with the title: "On a point in the physiology of the nervous system not thoroughly investigated; and a peculiar form of paralysis." After asserting the connection of this form with tabes dorsalis, he proceeds as follows:—

"General sensibility is not deficient nor muscular contractility, but the perception of movement. . . . . The gait of these persons is peculiar; the incongruous movements which their limbs execute are not met with in ordi-

[1] Anat. Pathol. (1837), livraison xxxii.

nary paralysis. . . . As they have not the perception of their movements they supply its want by the exercise of the other senses, and especially the sight; hence it is difficult or even impossible for them to walk in the dark."[1]

Next in order of time comes the celebrated case of Mr. Hanley, in which, without evident external cause, the loss of motion in the lower limbs gradually, and after several years, became complete without the sensibility being impaired. After death the substance of the cord in its posterior half, and in its entire length, from the pons to its lower end, had turned of a dark brown colour, and was extremely soft and tenacious; all the other parts were healthy.[2] This was one of the first cases to shake confidence in the doctrine that the posterior columns are conductors of sensitive impressions.

In 1843, it may be mentioned, Canstatt[3] gave a complete history of the imperfect paralysis of the lower limbs connected with tabes dorsalis, and attributed the loss of power to *atrophy* of the spinal marrow, and especially of its lower end; but he did not distinguish the columns of the organ which were especially the seat of change.

Of all who earliest described this disease Wunderlich presented its characteristic trait most plainly in his work on Practice of Medicine in 1853–4. He particularly calls attention to the fact that there is not so much a loss of power in the limbs, as a peculiar insecurity and uncertainty in their function. "The patient," he remarks, "even when he has long been unable to take a steady step, can yet stamp vigorously on the floor, and in bed move his limbs freely without the least fatigue."

It is unnecessary to pursue this line of discovery any further, since enough of it has been examined to prove that the affection we have been studying was long ago described by its symptoms and lesions. To interpret them, however, required the light which Dr. Brown-Séquard has thrown upon them from the combined sources of experimental physiology and clinical observation. It is undoubtedly true, as that gentleman asserts, that locomotor ataxia is found in numerous spinal affections; for quite probably there are more instances in which the spinal marrow is injured in several of its divisions, than there are cases in which the lesion is strictly confined to its posterior columns. The latter must, however, be taken as the simplest and typical cases, the standards with which those are to be compared in which locomotor ataxia occurs as one only of an aggregate of symptoms. A. S.

[1] Quoted by Axenfeld, Arch. Gén., Aug. 1863, p. 214.
[2] Med.-Chir. Trans., xxiii. 80, 1840.
[3] Handbuch der Med. Klinik., 2d ed., iii. 207.